

Field Epidemiologic Studies of Populations Exposed to Waste Dumps

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Epidemiologic studies are required for assessing health risks related to toxic waste exposure. Since the settings in which such studies must be performed are extremely diverse, epidemiologic approaches must be versatile. For any particular study, three fundamental requirements are to assess what toxic materials are present, understand how human exposure may occur, and objectively measure possible biologic effects. In assessing links between exposure and disease, epidemiologists must be particularly aware of: expected disease frequencies in relation to the size of populations studied, implications of long or varied disease latencies for study design and competing causes of disease and associated confounding variables. These concepts are illustrated by discussion of epidemiologic studies related to the Love Canal toxic waste dump site in Niagara Falls, NY.

A worrisome by-product of modern industrial society is the massive problem of toxic waste disposal. The potential for such wastes to cause health damage in exposed human populations requires the performance of epidemiologic investigations to assess relationships between toxic exposure and possible health consequences, clinical or subclinical. Although such investigations are conceptually simple and involve straightforward concepts of cause and effect, their success entails not only complex and expensive technical assessment of exposure, but also close attention to the statistical and methodologic requirements of effective epidemiologic design. This paper concerns these latter requirements and presents as illustrations several toxic waste health studies recently conducted in the United States (Table 1).

A general feature of such studies is the sheer diversity of situations in which toxic wastes can be involved. No two situations, and therefore no two studies, are exactly the same. Differences, in fact, can be extreme, ranging from the common stereotype of a drum-filled dump site to the widespread dispersal of waste material. The five toxic waste situations described in Table 1 illustrate this diver-

sity, both in waste materials and in exposure settings. The first two listed (the Love Canal and the Melvin Wade dumps) constitute discrete and relatively organized dumping. The wastes consisted mostly of organic materials, with human contact being by diverse routes at Love Canal (1) and by direct contact, fire, and explosion at the Wade site (unpublished data, Centers for Disease Control, Atlanta, GA, 1979). In Woburn, MA, toxic chemical wastes remained from past industries involved in tanning and in the production of arsenical pesticides (2). Potential exposure to both organic and inorganic materials may have occurred by both direct and waterborne routes (from contaminated local wells that provided drinking water). In Triana, AL, waste material from the manufacture of DDT pesticides was dumped in a local stream (3,4). High levels of DDT congeners developed in fish in the stream and in the local rural population consuming the fish. In Bloomington, IN, a local manufacturer of electric equipment discharged wastes containing polychlorinated biphenyls (PCB) into the city sewer system (5). PCB-containing sludge from the sewage treatment plant was then used for organic gardening by members of the local community.

In the first three settings, since levels of chemicals in exposed persons could not be measured directly, potential health effects had to be assessed indirectly. At the Love Canal, this took the form of extensive questionnaire surveys as well as a review

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Table 1. Recent situations involving potential human exposure to potentially toxic waste materials.

Location of site	Toxic materials involved	Physical condition	Principal routes of potential human exposure
Love Canal dump, Niagara Falls, NY	Largely hydrocarbon residues from pesticide production	Inactive land-fill in residential area	Direct, airborne, and waterborne contacts
Melvin Wade dump, Chester, PA	Diverse organic chemicals	Surface collection of waste in drums in urban setting	Direct contact, explosion, and fire
Woburn, MA	Arsenic compounds, heavy metals, organic chemicals	Abandoned waste lagoon with multiple surface dumps	Direct and waterborne contacts
Triana, AL	DDT and related compounds	Industrial waste dumped in a rural stream	Food chain (fish)
Bloomington, IN	Polychlorinated biphenyls (PCB)	Industrial waste contaminating municipal sewage used for garden manure	Direct contact and possibly food chain

of local cancer incidence patterns in relation to residential proximity to the dump site. At the Wade site, where residents' contact with the dump was less direct and where concern focused on the exposure of firemen during a past explosion and fire on the site, health studies were limited to a brief questionnaire survey of nearby residents and a review of medical records for affected workers. In Woburn, community concern regarding local increases in cancer mortality rates, and especially a local time-place cluster of childhood leukemia cases, caused initial epidemiologic studies to focus on a review of local cancer incidence and on questionnaire case-control studies for selected malignancies. No clear etiologic relationships were defined.

In the other two settings, Triana and Bloomington, levels of persistent chemicals (DDT/DDE, PCB) in exposed persons could be measured. Elevated levels were found in the first instance, but in the second it appeared that contact with PCB-contaminated sludge used for gardening purposes did not increase body levels of the chemical, although increased levels were seen in workers at the electrical manufacturing plant. Neither setting has been clearly associated with clinical illness.

Requirements of Epidemiologic Studies

Epidemiologic studies in settings such as these, regardless of their diversity, consist of three fundamental phases: (1) assessing the nature of toxic materials present, (2) understanding how human exposure to these toxins might occur and (3) evaluating potential biologic effects. Adequate information in each of these phases is critical for the success of any epidemiologic study.

Toxins

The first step in any toxic waste study is to determine what toxic materials are present and in what amounts. Since most toxic waste situations involve the dumping of diverse materials, problems of expensive technical methodology can be formidable. However, without an adequate inventory of what particular chemicals are present, in what quantities, and under what physical conditions, it is premature—if not impossible—to design adequate epidemiologic studies. If only low concentrations or small amounts of toxins are present, there may be insufficient reason to proceed with studies. The decision of whether or not to proceed is not always as simple as it may seem, since public concern can be compelling even in the absence of a confirmed toxic exposure.

Despite such pressures, it is important to realize that conducting even a simple survey of health effects is perilous if one lacks information about toxins and exposure, since few—if any—measurable health effects are sufficiently specific for exposure to particular toxins or groups of toxins to be surrogates for directly measuring toxic exposure.

Exposure

After assessing the nature and quantities of toxins present, one must evaluate their potential for human exposure. Despite the presence of clearly toxic materials, human exposure may not have occurred or may be only remotely possible. Before exposure can be evaluated, the modes of exposure (for example, direct contact, contaminated water or contaminated air) must be determined, and the size of human populations potentially exposed and their degree of proximity to the toxic materials must be

defined. This can be a complex issue, particularly if exposure, as often is the case, involves contaminated aquifers used for drinking water, thus exposing populations otherwise remote from the toxic waste source.

One must also consider the nature of chemicals involved, since some chemicals expose persons only transiently and others are stored in tissue, thus presenting a potential for continuous and cumulative exposure long after an active external exposure has ceased. Exposure to chemicals which persist in tissue, of course, provides much greater opportunity for productive epidemiologic study (the studies in Triana and Bloomington are examples) than exposure to transient agents, since many studies are undertaken long after exposure has occurred. In such settings, exposure can be judged objectively by measuring levels of persistent toxins in tissue (for example, levels of polychlorinated or polybrominated biphenyls in serum or fat), an option not available for transient toxins.

Biologic Effect

The prime objective in epidemiologic studies is to associate particular exposures with potential biologic effects and thus to define cause-effect relationships. Since this process is by nature an indirect assessment of etiology, it is highly dependent on the precision and specificity of observations recorded both for potential exposure and for potential biologic effect. It gains particular power if dose-response relationships can be shown, that is, if increasing levels of exposure are associated with increasing frequency of the biologic effect.

To assess the presence or absence of etiologic relationships, the epidemiologist must be aware of three particular difficulties which can severely limit the power of an epidemiologic investigation (Table 2). The first of these involves the size of the population needed for study to demonstrate a given health effect with a given degree of power. This depends

both on the degree of exposure involved (dose) and on the expected baseline frequency of the particular health effect. If the health outcome to be assessed is relatively rare, the population to be studied will need to be relatively large.

The second difficulty is that of long or variable latency. Particularly at lower levels of toxic exposure, health effects such as cancer cannot be observed (if in fact they are caused by the particular exposure) until years later. This requires that the study design either allow for long-term health follow-up or seek some subclinical biologic marker which can predict eventual cancer risk. Since no such markers have been developed (and their development involves a formidable array of methodologic obstacles), one is left either with prospects of long-continued study or with studies of current cancer occurrence in the instance where exposure has already existed over a span of years.

The third issue is that of competing causes, or expressed differently, the clinical nonspecificity of biologic effects under study. To the epidemiologist this means adjustment for potential confounding factors in a study. Since, as mentioned above, it is exceedingly unusual for a particular biologic effect to be the specific and exclusive result of particular toxic exposure, study design must allow for data to be collected about other exposures that might also give rise to the effect and about variables which might indirectly reflect degree of exposure to any or all such risk factors. In practical terms, this means collecting data regarding past occupational exposures, personal exposures such as cigarette smoking or use of alcohol or drugs, and personal characteristics such as sex, race, age, and socioeconomic status which may predict levels of risk for specific health outcomes. The more variables a study addresses, of course, the more complex its eventual analysis becomes, and the greater the size of the population needed for adequately assessing health effects.

These various principles may best be illustrated by a brief account of the Love Canal toxic waste

Table 2. Epidemiologic issues fundamental to evaluating potential toxic waste health effects.

Epidemiologic issue	Impact on epidemiologic study
Expected baseline frequency of specific health effects	Relatively low expected frequency requires relatively large population for study, especially to detect small increases in risk
Latency period	Long latency may require periodic or continuous long-term population follow-up
Multiple causative factors (clinical nonspecificity)	Since particular health effects are not often specific for particular toxic exposures, data must be collected and analyzed regarding multiple hosts and exposures

problem and the process by which specific epidemiologic studies have been developed and interpreted in that particular setting.

Epidemiologic Studies of Love Canal

An abandoned 16-acre ditch was used from 1942 to 1953 as a dump site, primarily for industrial chemical waste. When the dumping was discontinued, the site contained about 22,000 tons of chemicals, largely related to past production by the Hooker Chemical Company of the pesticide lindane (hexachlorocyclohexane). In 1953 the Canal was covered with a clay cap. The area later became the site of progressing suburban residential development, including the construction of homes and an elementary school directly on the borders of the Canal. The clay cap was apparently disturbed during the construction of streets, sewers, and utility conduits across the Canal. By the mid-1970s, chemical seepage was evident at the Canal's surface. Chemicals had spread through the soil into adjoining residential lots, facilitated by seepage of surface water into the Canal. In the spring and summer of 1978, as engineering plans developed to correct this problem, epidemiologic studies were begun to assess potential relationships between health patterns in the community and exposure to chemicals from the Canal.

The population at risk was first defined as persons living in the first two "rings" of homes adjoining the east and west sides of the Canal (99 dwellings, about 300 persons). Attention later extended to the general nearby community of about 4000 persons, some living at sites where natural surface drainage patterns, antedating community development, might conceivably facilitate trans-soil seepage of chemicals. To define actual exposure levels in these populations, particularly adjacent to the Canal, the New York State Department of Health (NYSDH) performed extensive environmental chemical tests (soil, water, and indoor and outdoor air). Potential oncogenic, mutagenic and teratogenic chemicals (benzene, chlorobenzene and others) were identified in the Canal. Low-level contamination with such chemicals was also clearly evident at various sites in the first two rings of homes, but only in trace amounts elsewhere (including homes along natural drainage paths) (1). The results of further environmental tests are still being analyzed.

NYSDH collected health information through an interview survey of local residents, backed by a review of medical records (6,7). Information was sought on a wide range of conditions. No unusual

patterns were noted, except possibly with respect to certain reproductive effects. Special attention was given to reproductive outcomes, since latency considerations and limited sample size precluded immediate conclusions regarding possible low-dose oncogenic effects, and since potential fetal sensitivity to toxic effects encouraged a focus on pregnancy.

A summary of pregnancy outcomes (abortion, birth defects and low birth weight) is shown in Table 3, both for women living close to the Canal (first two rings) and for women living elsewhere in the Canal area, some where risk of Canal seepage was possibly increased. No clear increased risk was seen for any of the three pregnancy outcomes in women living next to the Canal. Although an increased frequency of low birth weight in women from homes with possible seepage risk suggested a toxic effect, the finding does not correspond well with NYSDH environmental test results.

In a later epidemiologic study, NYSDH examined cancer incidence patterns in the Love Canal area (8). Rates in the census tract containing the Love Canal were compared with rates in other tracts in Niagara Falls. No consistent differences were found, although some lung cancer rates in women were elevated. Specifically, no increases over time were seen for liver tumors or for hematopoietic malignancies (leukemia, lymphoma).

Comment

The several epidemiologic limitations described earlier can be clearly seen in the results of the investigations performed at Love Canal. Since the health problems studied are rare, the numbers of observed cancer cases or adverse pregnancy outcomes were small, despite their occurrence in what might be considered a highly populated area. The actual exposed population, of course, may have been somewhat smaller than those populations defined for epidemiologic study (particularly the full Love Canal census tract examined for cancer incidence) if one accepts the NYSDH environmental test results and assumes that the levels of chemicals detected in 1978 were not greatly exceeded in previous years.

This limitation in population size and in frequency of illness outcomes is accentuated, of course, by the additional problems of long latency (in the case of the cancer study) and multiple causative factors. In analysis of pregnancy outcomes, close attention was given to adjustment for birth order, maternal age, and maternal history of smoking. However, since abortions, birth defects, and low birth weight can all result from many causes (or interacting

Table 3. Pregnancy outcomes in women residing as of June 1978 in the Love Canal area, Niagara Falls, NY.^a

Pregnancy outcome	Place of residence			
	Next to the canal ^b	East of the canal		Comparison area north of the canal
		Near natural drainage swales	Not near natural drainage swales	
Total pregnancies	79	108	164	125
Abortions				
Number	15	25	21	11
%	19.0	23.1	12.8	8.8
Live births	65	85	144	110
Birth defects				
Number	4	10	7	8
%	6.2	12.0	4.9	7.3
Low birth weight (< 2500 g)				
Number	1	13	10	3
%	1.5	15.7	6.9	2.7

^aAdapted from NYSDH data (?).^bResidences on 97th and 99th Streets which border the Canal site on east and west.

causes), interpretation of the Love Canal findings remains difficult. This is particularly so for abortions and birth defects, since one must reckon with a considerable likelihood of bias in observer recall.

Since common and cause-specific clinical markers for potential toxic waste health effects are lacking, further studies of exposed populations will need to focus both on the sustained long-term follow-up of health effects and, at the same time, on the development of subclinical indicators for precisely predicting risk of eventual illness. Neither approach is simple. Long-term population follow-up studies entail not only considerable expense and logistic commitment but also serious ethical issues. Although subclinical markers, such as cytogenetic or mutagenic tests, have received much attention in recent years, they are not ready for field application. A vivid illustration of this gap was the unsuccessful attempt to relate cytogenetic findings in Love Canal residents to concrete clinical predictions (9). Until such cellular or subcellular tests can be clearly correlated with specific disease risks and with other biologic markers, their use in epidemiologic studies remains limited.

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